

Abstract - Exploring the impact of dietary patterns and ultra-processed food consumption on the severity of biopsy-proven steatotic liver disease

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Introduction

The consumption of ultra-processed foods (UPF) has significantly increased in recent years, raising concerns due to its positive association with obesity and diabetes. However, the relationship between ultra-processed foods and the severity of steatotic liver disease (SLD) has been less studied. Nonetheless, studies have indicated that both moderate (vs. low) and high (vs. low) consumption of UPF significantly elevate the risk of metabolic dysfunction-associated steatotic liver disease (MASLD). No study has been conducted on UPF consumption in alcohol-related liver disease (ALD).

Aim

Our aim is to evaluate nutritional intake and UPF consumption in SLD.

Methods

Patients with histologically confirmed hepatic steatosis (MASLD or ALD) were prospectively included, excluding those with MetALD. Anthropometric and biological data were collected. Dietary intake was assessed through a 24-hour recall, and the NOVA classification was employed to quantify ultra-processed food (UPF) consumption in grams. UPF consumption data were compared with those of the general population available in the literature (Vandevijvere et al., 2019). Finally, the severity of liver phenotype was histologically evaluated using the Beaujon score (SAF).

Results

Dietary intake and consumption of ultra-processed and processed foods were assessed in 62 SLD patients (46 with MASLD and 16 with ALD). The mean age of MASLD and ALD patients was 54 and 52 years respectively (NS). MASLD patients exhibited significantly higher mean body mass index (BMI) and abdominal circumference compared to ALD patients (35 vs. 22 kg/m², $p = 0.0001$; 117 vs 91 cm; $p = 0.0001$). Biological data showed a mean GGT level of 64 U/L in MASLD patients and 590 U/L in ALD patients ($p = 0.0001$), a mean HDL-C level of 43 mg/dl in MASLD patients and 89 mg/dl in ALD patients ($p = 0.0001$), and mean triglyceride levels of 181 mg/dl in MASLD and 196 mg/dl in ALD (NS). Both MASLD and ALD patients presented a mean moderate degree of steatosis, histologically assessed as grade 2. Among the 62 histologically evaluated patients, 3 were classified as F0 (4.9%), 12 as F1 (19.7%), 28 as F2 (45.9%), 17 as F3 (27.9%), and 1 as F4 (1.6%). One patient could not be evaluated due to the biopsy's size. Although MASLD patients had a significantly higher BMI than ALD patients, the energy intake of MASLD patients was significantly lower than that of ALD patients (1806 vs. 2716 kcal/day; $p = 0.0003$). In terms of nutritional intake, MASLD patients consumed more fats (73 vs. 53 g/day; $p = 0.050$) and fibers (17 vs. 8 g/day; $p = 0.0003$) than ALD patients. Our results also indicate that MASLD patients consume more ultra-processed and processed foods than the general population (ultra-processed foods: 40 vs. 35% of total energy intake (TEI); processed foods: 24 vs. 13% of TEI). When alcohol consumption is considered, ALD patients consume more processed foods than MASLD patients (2628 vs. 196 g/day; $p = 0.0001$). No impact of UPFs on the severity of the hepatic phenotype in terms of steatosis, inflammatory activity, and fibrosis is evident.

Conclusions

ALD patients consume more calories than MASLD patients, despite being significantly thinner. The difference in energy intake between patients with MASLD and those with ALD is mainly attributed to the caloric contribution of alcohol consumption, which is also responsible for the greater consumption of processed foods. Despite an increased consumption of UPF in MASLD patients compared to the general population, this dietary habit does not appear to significantly influence the severity of the hepatic phenotype. This observation underscores the multifactorial complexity of the disease.

